Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	14	anaphylatoxin adj c3a adj receptor	US-PGPUB; USPAT; DERWENT	OR	OFF	2005/02/07 12:13

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS, LIFESCI' ENTERED AT 12:27:33 ON 07 FEB 2005

L1	1141	S ANAPHYLATOXIN (A) C3A
L2	101	S L1 (A) RECEPTOR
L3	43	S L2 AND (KNOCKOUT OR DISRUPT OR MOUSE)
L4	23	S L3 NOT PY>2000
L5	10	DUP REM L4 (13 DUPLICATES REMOVED)
L6	337	S C3AR
L7	112	S L6 AND (KNOCKOUT OR DISRUPT? OR MOUSE)
L8	52	S L7 NOT PY>2000
L9	21	DUP REM L8 (31 DUPLICATES REMOVED)
L10	16	S L9 NOT L5

AN 2001059574 MEDLINE
DN PubMed ID: 11067891
TI Cutting edge: targeted **disruption** of the C3a receptor gene demonstrates a novel protective anti-inflammatory role for C3a in endotoxin-shock.
AU Kildsgaard J; Hollmann T J; Matthews K W; Bian K; Murad F; Wetsel R A
CS Institute of Molecular Medicine for the Prevention of Human Diseases, University of Texas-Houston, Houston, TX 77030, USA.
NC AI25011 (NIAID)
SO Journal of immunology (Baltimore, Md. : 1950), (2000 Nov 15) 165 (10) 5406-9.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200012
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001222
AB The complement anaphylatoxin C3a, on binding the **C3aR**, mediates numerous proinflammatory activities. In addition, recent in vitro studies with C3a have implicated **C3aR** as a possible anti-inflammatory receptor. Because of its possible dual role in modulating the inflammatory response, it is uncertain whether **C3aR** contributes to the pathogenesis of endotoxin shock. Here, the targeted-**disruption** of the **C3aR** in **mice** is reported.
These **mice** exhibit an enhanced lethality to endotoxin shock with a pronounced gene dosage effect. In addition, the plasma concentration of IL-1beta was significantly elevated in the **C3aR**(-/-) **mice** compared with their littermates following LPS challenge.
These findings demonstrate an important protective role for the **C3aR** in endotoxin shock and indicate that, in addition to its traditionally accepted functions in mediating inflammation, the **C3aR** also acts in vivo as an anti-inflammatory receptor by attenuating LPS-induced proinflammatory cytokine production.

TI Molecular cloning of two isoforms of the guinea pig **C3a anaphylatoxin receptor**: alternative splicing in the large extracellular loop.

AU Fukuoka Y; Ember J A; Hugli T E
CS Department of Immunology, The Scripps Research Institute, La Jolla, CA 92037, USA.

NC AI41670 (NIAID)
DE10992 (NIDCR)
SO Journal of immunology (Baltimore, Md. : 1950), (1998 Sep 15) 161 (6) 2977-84.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
OS GENBANK-U86378
EM 199810
ED Entered STN: 19981020
Last Updated on STN: 20000303
Entered Medline: 19981006

AB The anaphylatoxin C3a is released from C3 during complement activation. C3a is a potent spasmogen and has recently been described as an eosinophil and mast cell chemotactic factor that mediates a number of inflammatory reactions. Previously, we demonstrated the presence of a specific C3a receptor (C3aR) on guinea pig platelets. We report here the isolation of cDNA clones encoding for two isoforms of guinea pig C3aR (gpC3aR). Hydropathy analysis of the deduced amino acid sequence of both gpC3aR clones indicated seven transmembrane domains with a large extracellular (EC) loop between the fourth and fifth transmembrane domains, which is a known characteristic of the human C3aR. Northern blot analysis revealed that the gpC3aR was abundantly expressed on macrophages and in the spleen. A comparison of the deduced amino acid sequence of the larger gpC3aR (gpC3aR-L) with the recently cloned human C3aR indicated a 59.5% identity. The deduced amino acid sequence of the second, smaller cDNA clone was identical with gpC3aR-L, except that it lacked 35 amino acids in the large EC loop. Our evidence indicates that alternative splicing occurred in the large EC loop that accounts for these two isoforms. L cells separately expressing one of these two isoforms of the gpC3aR showed similar high-affinity C3a binding. An RT-PCR analysis documented that both forms of the C3aR were expressed in a variety of guinea pig tissues. The cloning and expression of these two natural forms of gpC3aR cDNA indicated that the deletion of the 35-residue portion of the large EC loop of gpC3aR-L did not alter C3a binding.